WADC TECHNICAL REPORT 53-139
PART 1

THE PHYSIOLOGY OF SIMPLE TUMBLING

Part 1. Animal Studies

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WRIGHT AIR DEVELOPMENT CENTER

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FOREWORD

This report concerns the investigation of tumbling, one of the numerous problems encountered during escape from aircraft. The study was first proposed by T. C. Hill, Capt, USAF (MC) and R. S. Shaw, Capt, USAF (MC) in 1948, and later performed at the Aero Medical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, under the authority of RDO 695-61, "Escape from Aircraft" as sub-project R-695-61 A. The principal investigators for the phase dealing with animal experimentation were Robert Edelberg, Capt, USAF and Harold S. Weiss, Capt, USAF, assisted by Paul V. Charland,1st Lt, USAF and J.I. Rosenbaum, M. D..

The authors wish to acknowledge the valuable advice of numerous personnel of the Aero Medical Laboratory as well as the technical assistance of Paul G. Winquist, Capt, USAF (MC), Wallace E. Wendt, Capt, USAF (VC) and Mr. Walter Orlow.

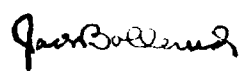
ABSTRACT

The tumbling that follows emergency escape from an aircraft by seat ejection or that occurs during prolonged free-fall poses a threat to the escaping crewman. Tumbling was simulated in the laboratory on a horizontal spin table using anesthetized dogs as subjects preliminary to human experimentation. The axis of rotation was through the heart or at various locations up to 20 cm caudad. The centrifugal forces proved effective in producing peripheral pooling with a consequent reduction in heart filling and cardiac output, as evidenced by the reduced pulse pressure and arteriovenous pressure difference. The decrease in perfusion pressure and the accompanying apnea was enough to produce hypoxia at speeds greater than 140 rpm, as evidenced by oral cyanosis. Concurrently, the elevated hydrostatic pressures were sufficient to produce hemorrhage in the extremities. A tachycardia or bradycardia may occur, depending on the location of the center of rotation. In general, pathology is less when the center of rotation is at the heart than when located at the more caudad positions, but circulation is less impaired as the center is moved caudad.

PUBLICATION REVIEW

This report has been reviewed and is approved.

FOR THE COMMANDER:



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SECTION I

INTRODUCTION

A. The Problem

Following emergency escape from aircraft, there often occurs a head over heels tumbling motion which has been observed to reach speeds of 180 rpm (1) and, on occasion, has been estimated to exceed 250 rpm (2). An example of this rotation may be seen in Figure 1 which is a composite photograph of a film strip taken during an actual flight. In this particular case the dummy and seat completed three revolutions before passing the tail, giving a tumbling rate in excess of 180 rpm. Another form of tumbling, generally in the nature of a flat spin, may develop during the long free fall following high altitude bailout. Rates of rotation of dummies as high as 160 rpm have been measured under these conditions (3). Calculations show that tumbling at rates between 180 and 240 rpm may produce forces at head level greater than 30 negative g, and thus could be a potential threat to the escaping crewman. Therefore, it is important to know the effects of such rotation on the body and to determine the limit of human tolerance to the forces produced.

Figure 1. Composite photograph of actual ejection from a B-47. Rotation is greater than 180 rpm.

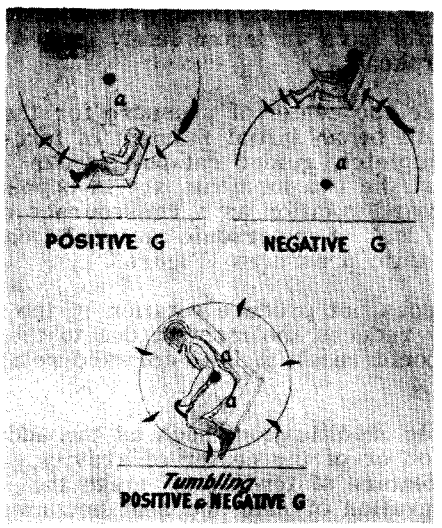

Figure 2. Mass movement of blood during various types of acceleration.

Unfortunately, simple extension of the existing knowledge of human tolerance to positive and negative accelerative stresses will not suffice in determining tolerance to tumbling (4) (5) (6) (7). If, with the aid of Figure 2, one visualizes the situation that exists during rotation about an axis through the body, it is apparent that the centrifugal forces will drive blood headward and footward simultaneously, producing both positive and negative-g conditions at the same time. Furthermore, in contrast to the uniform force fields generally considered in connection with positive and negative g, a steep gradient of g and hydrostatic pressure will exist within the body during tumbling, starting at zero at the center of rotation (C/R) and increasing rapidly outward. This report, then, describes the general physiological and pathological effects of tumbling prior to conducting similar studies on human beings.

B. The Physics of Tumbling

The tumbling which accompanies seat ejection occurs immediately after separation of the seat from the aircraft and is associated with the linear deceleration of the seat in the air stream. This deceleration may, at current aircraft speeds, reach 14 g and can be expected, in the case of ejection at 800 mph, to reach 54 g at sea level, or 29 g at 20,000 feet (8). Both tumbling and linear deceleration are virtually over in three to five seconds at these altitudes (1) (2). Since the high drag forces are always superimposed on any tumbling which follows ejection, it is apparent that the investigation of simple rotation (i.e., with no superimposed g-field) cannot furnish information which is directly applicable to this situation. However, the study of simple tumbling is a necessary preliminary to the investigation of combined acceleration, In the case of a spontaneous flat spin occuring during long free fall, the results of experiments on simple rotation may be applied directly, although there could be a superimposed linear deceleration caused by increasing air resistance during the fall (9).

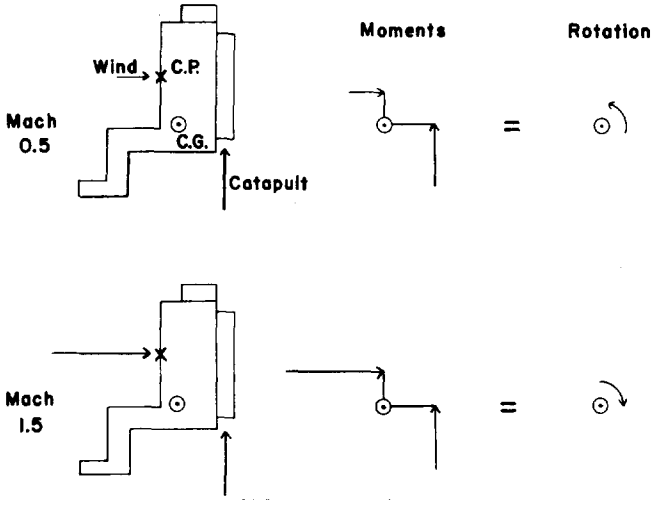

Figure 3. Interaction of dynamic air pressure and catapult force in the generation of tumbling.

While it is not within the scope of this report to discuss at length the origin of tumbling following ejection, a few word of explanation may be profitably offered. Tumbling is due to a moment of force generated either by the catapult or by wind pressure (Figure 3). In the existing USAF ejection seats, the catapult is located aft of the center of gravity. This causes the seat to bend forward on the track as it travels upward and, as the seat separates from the track, a head-forward moment results (1). In the case of a completely guided stroke, i.e., one in which the upward acceleration would end before the seat left the track, the moment would disappear before separation and there should be no forward rotation. However, current USAF upward ejection seats have an incompletely guided stroke.

With regard to dynamic pressure in the air stream, since the center of pressure for the seats presently in use is above the center of gravity, there will be an initial moment of force tending to cause a backward rotation. In the case of an incompletely guided catapult stroke followed immediately by exposure to wind blast, it is seen that the two moments act in opposition. The usual type of upward ejection results in a head-forward rotation as a consequence of the dominance of the catapult moment at current aircraft speeds. At supersonic speeds, a backward rotation may be developed as a result of the higher dynamic pressures (Figure 3).

Contrary to what might be assumed, downward ejection does not produce rotation in the opposite directions. A more completely guided stroke greatly reduces the moment due to the catapult. The wind blast, then, acting on the foot end first, could result in head-forward rotation at all times.

It is possible to eliminate rotation of the seat by suitable modification, such as the addition of fins, use of drogue chutes, or a shift of the catapult position or of the center of gravity. However, all of these steps carry with them the undesirable feature of tending to make the seat heavier, more complicated or unwieldy. In addition, separation of man and seat becomes more difficult in the absence of tumbling. Finally, it is conceivable that tumbling may reduce

the vascular damage caused by the strong decelerative forces occuring in the air stream (2) (10). Negative-g studies show that rupture of the vessels occurs if a given pressure acts for a few seconds. If the inertia of the blood is such that it cannot be displaced in response to very short periods of high intensity g-loads, little rupture of vessels would be expected when the duration of force in any given direction is sufficiently shortened by rapid tumbling.

C. Historical Background

Prior to 1945, both the Germans and British were aware that tumbling might be a troublesome corollary of the ejection method of escape from aircraft. British calculations and experiments indicated a potential rate of tumbling of 30 to 90 rpm (11 a) (11 b) (12) whereas the Germans arrived at a maximum rate of only 30 rpm (11 a). Since Wieshofer was reported to have spun human subjects at 90 rpm with the center of rotation at the center of gravity without ill effects (11 c), the Germans concluded that simple tumbling was not a serious problem and concentrated most of their efforts on other aspects of ejection. They did, however, consider the possibility of a dangerous situation developing if the spin oriented the subject in such a way that deceleration in the air stream produced high negative-g forces (11 d). Tests were conducted on ejection seats that had an increased surface area in the upper portion to insure that the subject would not be initially oriented in a negative-g position (11 e). Apparently even this consideration was abandoned on the advice of Weishofer that the maximum duration of the anticipated negative g was too short to be harmful (11 f).

However, to British thinking, the consequence of tumbling could possibly be severe enough to cause incapacitation. Jasper (13), in the course of a study on positive and negative g, carried out a number of tests on monkeys in which the axis of rotation was through the body with the center of rotation varying from the neck to the heart. Top speed attainable was 120 rpm. On the basis of the electroencephalographic patterns, they concluded that the effects were similar to straight positive g of the same magnitude. Unfortunately, these tests were not sufficiently extended to cover the range of conditions likely to be encountered in tumbling. In the absence of more specific information on human tolerance to tumbling, the British chose to stabilize the seat by means of a guided catapult stroke and a drogue chute (14), a technique also adopted by the U. S. Navy. However, free fall of subjects in ejections fron 40,000 feet (1950) showed that drogue chutes were not without disadvantage. During these falls the drogue chute oriented the seat in such a position that an uncontrollable spin occured (15).

Investigators in the United States soon confirmed the British and German reports that tumbling may occur following ejection. The need to further explore this type of stress on the body was recognized by the Aero Medical Laboratory in 1946 (16). At this time, the USAF also initiated studies on stabilization of the seat by means of parachutes (9). Continued investigation of the ejection problem brought forth a variety of results and opinions insofar as stability of the seat was concerned; some are enumerated here:

(a) Rates and durations of tumbling encountered in the 1946 flights made at 290 mph IAS and 12,000 ft. were considered insufficient to produce serious negative g forces (17).

(b) Ground tests in 1947 indicated a potential tumbling rate of 128 rpm but only 75 rpm was measured in the 1948 flight tests (18).

(c) In 1948 the Douglas Aircraft Company developed a seat which was said not to tumble or twist during actual ejections (19).

(d) In connection with escape from the XS-2 aircraft, studies of the falling characteristics of a capsule enclosing the seat were recommended because of the possibility that spin would hamper bailout and foul chute suspension lines (20).

(e) Decelerative forces in the region of 65 to 160 g, in addition to superimposed tumbling, were predicted for capsule ejections(21).

(f) Stabilization of ejection seats by means of guide surface parachutes were again suggested in 1948 (22). Drogue chutes were recommended primarily to insure positive separation of man and seat.

(g) In 1949, the USAF measured combined accelerations in the neighborhood of 11 negative g for 1/10 second in actual ejections at 349 knots (23). However, reports of a series of live ejections at even higher speeds indicated no interference with normal escape procedure despite rotation of the seat (24). Angular accelerations up to 333 radians/sec^2 associated with 16.5 negative g were recorded in subsequent tests (25). Forward rotation of the seat could be stopped by means of flaps, but this was considered unnecessary since the accelerations were not estimated to be above the tolerance limits.

Progress in capsular escape from aircraft stimulated continued interest in tumbling, especially since flight in the range of 1500 mph and 100,000 feet altitude was contemplated (26). Goodyear Aircraft engineers thought stabilization of their capsule might be necessary (27) and, in fact, dynamic tests on models (1951) confirmed the expectation that both forward and backward pitching moments might develop (28). On the other hand, Douglas Aircraft engineers indicated that the detached nose section of the D-558 or D-558-2 would not auto-rotate although small scale models had exhibited potentially high spinning and tumbling rates (29).

On the basis of the simulated and live ejection tests reviewed here, it cannot be definitely stated that the forces generated in connection with tumbling have ever exceeded human tolerance. On the other hand, the possible development of a dangerous condition has not been eliminated, especially as the performance of the aircraft improves. Further, it would seem that more tumbling is encountered during actual emergency use of ejection seats than in the experimental situations. Analysis of 58 successful emergency ejections from F-84 and F-86 aircraft shows that approximately 25 % of the pilots experienced "severe" tumbling (30). Temporary blackout followed ejection in a few cases but cannot be attributed to any one cause. Despite a wide range of conditions under which ejections have taken place (1000 to 20,000 feet altitude, 140 to 500 knots, dives, spins, climbs, etc.,) no obvious correlation of the severity of tumbling with altitude, speed or attitude of the plane is evident. There are a number of unexplained fatalities following emergency use of the ejection seat (those in which the body has struck the ground still strapped in the seat). These cannot be attributed to low altitude at the time of escape. It has been suggested that g forces strong enough to produce unconsciousness may have been a contributing factor (31).

In 1948, the Aero Medical Laboratory, recognizing the lack of concrete experimental data on the effects of tumbling, proposed a study of the problem (32). Prior to that time, the only physiological data available appears to have been the previously mentioned German report (11), and studies by Jasper and Cipriani (13). More recently, portions of the work by Lombard et al (33) on combined tumbling and linear acceleration have become available. As summarized in a U. S. Navy report (2), Lombard and his associates found goats capable of surviving 72 rpm and five g, and that humans can withstand 40 rpm and two g without experiencing dizziness. Rotation about the heart was found to be the most tolerable.

The animal experiments on simple tumbling, which form the basis of this report, began in 1951 and have been published, in part, in abstract form (34) (35).

D. Theoretical Physiological Analysis

A preliminary discussion of the general effects of tumbling will be of value in understanding the experimental results that follow. For the sake of simplicity, the hemodynamic and hydrostatic effects to be expected from simple rotation will be considered for the case in which the center of rotation is through the heart. While it may seem that the centrifugal forces directed away from the heart would directly oppose the return of blood to the heart,

such a view is inaccurate. The movement of blood may be regarded as the result of a pressure gradient along the line of flow. When centrifugal force is applied, as in the above situation, an increment of pressure is produced in both the venous and arterial sides of the circulatory system. In itself, this will not affect the pressure gradient and flow should continue unabated as long as cardiac output is maintained and thus, we should expect tumbling to have no affect on the circulation. However, in the case of a mammal with its highly distensible venous bed, the increase in absolute pressure will produce an increase in peripheral bed volume which may act in the same way as an internal hemorrhage. This will result in a sufficient decrease in the volume of blood in the central veins to cause a fall in cardiac output. If this fall in cardiac output produces a pressure drop which is greater than the increase in hydrostatic pressure, there will then be a fall in arterial pressure rather than the expected rise.

When the center of rotation is moved caudad, the hydrostatic column from the center of rotation to the foot is shortened and a lesser degree of pooling in the legs and viscera can be expected. If this decrease in pooling is greater than the increase of pooling in the cephalic region, the caudad position may produce less interference with the circulation.. On the other hand, the negative-g symptoms (4) (5) (7) related to high intravascular pressures in the head region can be expected to increase. Conversely, movement of the center of rotation towards the head will increase the positive-g symptoms (4) (6) associated with pooling in the legs and viscera and lessen the pressure at head level. Thus, the circumstances under which a set of symptoms, or combination thereof, will become critical depends on the position of the center or rotation as well as the speed of rotation.

SECTION II

EQUIPMENT AND PROCEDURE

The basic piece of equipment used in the tumbling studies was a horizontal turntable, eight feet in diameter and capable of rotating at speeds between 15 and 200 rpm. Top speed is normally attained in four seconds, but may be attained in less if desired. The sheet metal surface of the table is stressed to support 200 lbs/sq ft. Two adjustable counterbalancing weights are suspended on the underside. Ten copper slip rings, arranged concentrically in a single plane, are used to transmit information to and from the table. (As originally designed, 12 troughs for use as mercury slip rings were available but proved impractical because of the splashing at high speeds). Motive power for the table is supplied by a 10 hp, 230 volt, shunt-wound DC motor.

Blood pressures were measured by means of Statham transducers (36) of appropriate range. To detect respiration rate, a device consisting of four strain gauges (37) cemented to a copper plate in a Wheatstone bridge circuit, was strapped around the animal's thorax. Deformation of the copper plate by respiratory movements unbalanced the bridge and produced a recordable signal. Needle electrodes coated with electrode paste and inserted under the skin of the fore and hind legs provided a standard electrocardiogram. Radial and tangential acceleration were obtained from the periphery of the table by means of Statham accelerometers while the speed of the table could be checked visually on a tachometer.

Recording equipment consisted of a Grass electroencephalograph (38) on which the animal's electrocardiogram, respiration, arterial pulse (for monitoring only) and the speed of the table could be recorded simultaneously. Blood pressures, acceleration and respiration were recorded on a Consolidated Oscillograph (39). Each measuring device used had an individual dry cell current source mounted on the table, allowing one side of the putput to be common, thus permitting maximum utilization of the available rings.

One hour prior to anesthesia, the dogs were morphinized (1/4 gr morphine sulphate in 2 cc saline, subcutaneously), mainly to evacuate the stomach and intestinal contents. Sodium pentabarbital (23 mg/kg, intravenously) was the conventional anesthetic although chloralose (1% solution in 0.6% saline) was used in a few cases. For a 15 kg dog, 200 cc of 1% chloralose was given initially, followed by 10 cc every hour. Deep anesthesia was employed during surgery, but anesthesia was maintained only at a medium to light level during the remainder of the experiment.

For surgery, the dog was placed on its side on a specially designed board which was also used to support the animal and anchor it to the spin table. The carotid and jugular veins of the upper side were exposed through a two inch incision, slightly lateral to the ventral midline of the neck. Similarly, the femoral artery and vein of the bottom hind leg were exposed through a medial incision in the thigh. All four vessels were then cannulated in one of three ways:

(1) End catheters of polyethylene tubing of appropriate diameter inserted one inch centrally into the vessel and tied in place.

(2) Polyethylene T-tubes through which flow was maintained in the cannulated vessels.

(3) Newly designed lateral catheters which clamped onto the vessel wall and permitted blood flow to continue.

All were filled with heparinized saline and connected to the pressure gauges through three-way stopcocks. The relative merits of the three catheters are compared in Appendix A.

After the catheters, electrocardiographic leads and respirometer were in place, the dog was braced as shown in Figure 4. A block of wood was placed between the teeth to prevent the animal from biting its tongue while under the g-forces. Despite prior administration of morphine, experience soon indicated the desirability of attaching a diaper arrangement to the animal in order to catch any previously unevacuated bowel contents. Also, the appearance of fairly frequent nasal hemorrhage led to the incorporation of the nose guard shown in the figure. Subsequently, a perforated plastic bag over the snout proved to be a better solution.

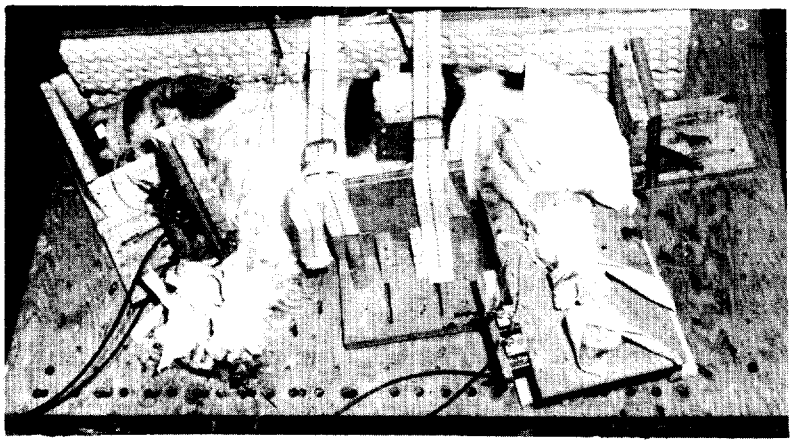

Figure 4. Dog in position on supporting platform in preparation for experimental tumbling.

The presence of "noise" in the electrical circuit was an annoying problem throughout the experiments, especially at higher speeds. Neither the room in which the spin table was located nor the wiring on the table had been adequately shielded in the original design and the copper slip ring -carbon brush combination was, in itself, particularly noisy. Ring noise could be reduced by sanding the rings with fine emery paper before a series of runs. The use of pairs of twisted leads for all circuits which moved with the table virtually eliminated any interference due to the cutting of stray magnetic fields by the spinning table. One source of noise in the blood pressure recordings was traced to the whipping and vibrating of the flexible catheters. This was reduced by shortening and tying down all exposed portions of the polyethylene tubing.

Two major types of runs were employed in the physiological phase of the investigation:

(1) G-runs - short exposures of 15 to 30 seconds over a wide range of speeds, mainly for the purpose of exploring the immediate reactions of the animal's circulatory system to tumbling.

(2) Time-runs - one to three minute exposures at speeds selected on the basis of the g-runs to bracket the region of critical physiological changes.

In a majority of the tests the center of rotation was at the heart, although positions cephalad and caudad from the heart were also investigated. The center of rotation was determined with a plumb bob and measurements were made from this point to various parts of the animal. Where one animal was used for a continuous series of runs, a sufficiently long interval was allowed between each run for the cardiovascular system to return to as near a normal condition as possible. At the conclusion of an experiment, animals that had been ligated on only one side were sutured and returned to the kennels for future use; others were sacrificed and a post mortem performed. Specialized procedures for certain phases of the work will be described in more detail as they are presented for discussion.

As an aid in analyzing blood pressure records, a chart of the fraction of pulse pressure to be added to diastolic pressure to obtain mean pressure was prepared from planimetric measurements of the various types of pulse curves encountered. The respiratory cycle was taken into account in determining mean pressure. In addition, a hydrostatic pressure correction was applied to convert gauge pressure to pressure at any nearby point, R_2, usually the point of insertion of the catheter. The formula referred to by Gauer (6) served as a basis for deriving the correction which, in the simplified form, appears as:

$$P_c = 4.115 \times 10^{-6} (R_2^2 - R_1^2) \quad rpm^2$$

where; P_c = Correction in mm Hg

R_2 = cm from C/R to point 2

R_1 = cm from C/R to gauge

SECTION III

RESULTS AND DISCUSSICN

A. Center of Rotation at the Heart

The general effects of rotation at high speed (e.g. 162 RPM) are shown in Figure 5. It is seen that the normally large arterio-venous pressure differences between the femoral artery and vein, and between the carotid artery and jugular vein, are quickly reduced to 10 or 15 mm Hg. In the one case this is caused by an increase in pressure in the femoral vein, in the other by a fall in pressure in the carotid artery. The rise in pressure in the femoral vein is readily explained by an increase in the hydrostatic pressure. It would be logical to expect that all pressures, including those in the femoral and carotid arteries, would likewise rise as the added hydrostatic pressures are imposed on the system. The fact that they do not must be attributed to a simultaneously reduced cardiac output. The loss of pulse pressure further substantiates this explanation. In the case of the femoral artery, the hydrostatic pressure compensates for the fall in cardiac output. In the case of the shorter column, from heart to neck, the hydrostatic pressure is insufficient to entirely counteract the fall in cardiac output and a fall in carotid arterial pressure results. In animals whose anatomical configuration is such that the hydrostatic column to the hind limb is shorter, arterial pressure may fall at both ends of the body (Figure 6). In either case, the explanation, based on the failure of cardiac output, is substantiated by the precipitous drop in arterial (particularly femoral) pressure when centrifugation ends. The sudden stop causes a decrease in hydrostatic pressure before the cardiac output returns to normal. As cardiac output builds up both the femoral and carotid arterial pressures rise to high levels. It is tentatively assumed, following the analysis offered in the Introduction, that the fall in cardiac output is a result of venous pooling in the viscera and extremities and that the return of cardiac output is associated with the shift of blood from the peripheral to the central veins.

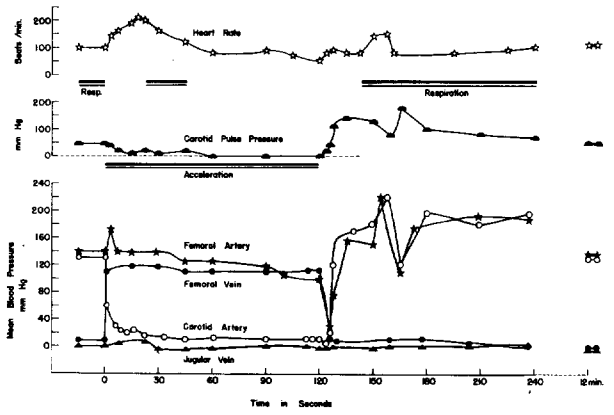

Figure 5 - Effects of spinning a dog about a center of rotation through the heart (162 RPM, 2 min.)

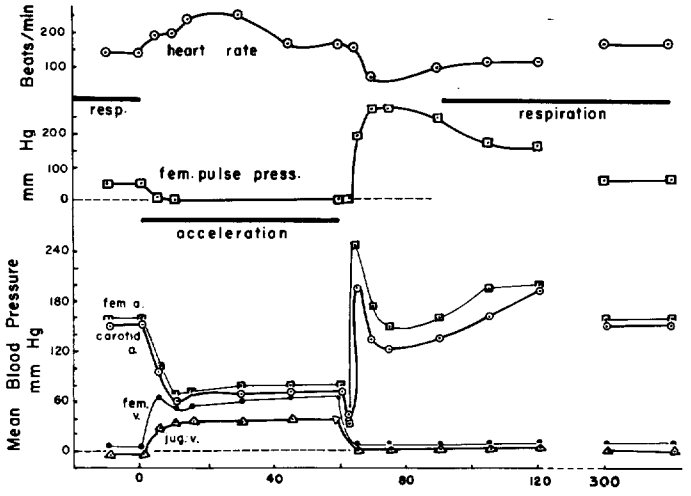

Figure 6 - Effects of spinning a dog about a center of rotation through the heart (171 RPM, 1 min.)

The curve obtained for heart rate from the electrocardiographic recording, indicates a tachycardia which reaches its peak of 220-250 beats per minute within 30 seconds after rotation begins. This is assumed to be a reflex response to the fall in carotid arterial pressure. After approximately 30 seconds of spin, the heart begins to slow down, despite the continued low carotid pressure. In view of the decreased blood flow, manifested by the low arterio-venous pressure difference and the loss of pulse, the decreasing heart rate may be the result of an insufficient coronary blood supply. This view is supported by the observation that at lower speeds (e.g. 107 rpm, Figure 7) flow persists for the duration of the run (note the sustained arterio-venous pressure differences and pulse pressure) and the tachycardia is maintained as well.

On cessation of centrifugation the return of the pulse and the arterio-venous pressure difference indicates that peripheral venous pooling has begun to reverse itself, heart filling is returning to normal, and cardiac output is being restored. From evidence to be presented later, it is believed that a widespread peripheral vasoconstriction develops during the run and persists for a few seconds after the run ends. This may, in part, explain the abnormally high arterial pressures which are achieved during the post-run period (Figures 5 and 6). Another contributing factor would be a more forceful contraction of the heart appearing as a consequence of the sympathetic response to hypoxia, hypercapnia and low carotid pressure existing during the run. The high post-run pressures subsequently invoke the carotid sinus reflex to produce a series of fluctuations in heart rate and blood pressure.

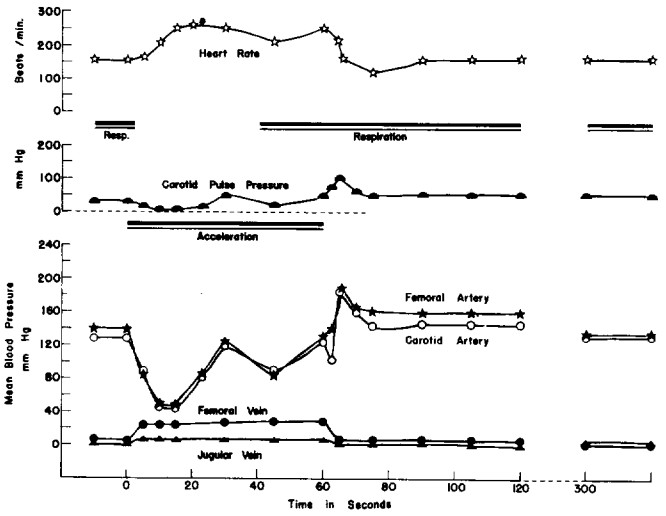

Figure 7 - Effect of spinning a dog about a center of rotation through the heart (107 rpm, 1 min.)

Pulse pressure falls off rapidly at the start of the run and all but disappears, reflecting the decrease in cardiac output already described. During the post-run period pulse pressure may become greater than 200 mm Hg in the initial phase. It is possible that these high pulse pressures may be due in part to the water hammer effect described by Maltby and Wiggers (40) (41). Evidence from a comparison of end and lateral-pressure measurements indicates that those pulse pressures may be as much as 36% too high (Appendix A).

Apnea develops at the start of the run and may even persist for several seconds after the run (Figures 5 and 6).

On the basis of two lines of evidence, this apnea is believed to be due to reflex activities, as well as to simple mechanical resistance to respiratory movements. For one thing, at lower speeds (Figure 7) respiration may return after an initial period of apnea indicating that respiratory movements are at least possible despite the g forces. Secondly, in experiments on dogs under light chloralose anesthesia (a drug which does not depress the respiratory reflexes as much as does nembutal (42)) higher speeds are required to prevent this return of respiration. For example, in a dog under chloralose anesthesia, return of respiration during the run still appeared at 161 rpm, while in the same dog under deep nembutal anesthesia, respiration reappeared only up to speeds of 110 rpm. Further, the persistance of apnea for several seconds following the end of the run would seem to require something more than a simple mechanical effect to explain it.

A possible explanation for the reflex cessation of breathing may be derived from knowledge that the viscera are pushed caudad under the stress of radial acceleration and in turn tend to pull the diaphragm downward. This would stimulate stretch receptors in the lungs, causing inhibition of the inspiratory center and stimulation of the expiratory center (43).

Examination of runs at lower speeds affords evidence for compensatory circulatory reflexes. Following the initial fall, the subsequent increase in mean arterial pressure and arterio-venous pressure difference during rotation at 107 rpm (Figure 7) cannot be attributed to tachycardia which has already reached 225 beats per minute with no concomitant recovery of the circulation. It is, rather, attributed to a reflex vasoconstriction. As in the case of the respiratory reflex, the circulatory reflexes are more manifest when chloralose is used instead of sodium pentabarbital. Figure 8 shows a comparison of the carotid pulse pressure for a single animal run at 107 rpm, first under chloralose anesthesia and then after an intravenous injection of nembutal. Results from a series of runs performed in a similar fashion at various speeds indicate that under light nembutal anesthesia and at a speed of approximately 140 rpm, the pulse disappears without returning while under chloralose it fails to reappear at about 165 rpm.

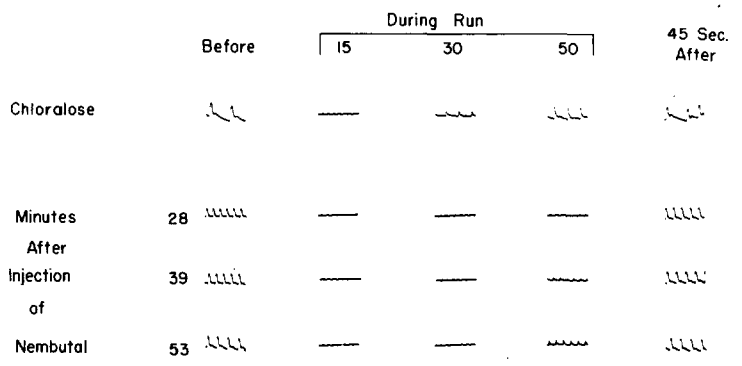

Figure 8 - Effect of anesthesia on return of pulse during tumbling.

The mechanism by which reflex activity may be stimulated to affect partial recovery of the disturbed circulation during the run can be briefly considered. During the initial period of the run venous pooling is increased (note the gradual increase in femoral venous pressure, Figure 7) hypoxia is developing, and carbon dioxide is accumulating (note apnea and decreased arterio-venous pressure difference, Figure 7). The role of hypoxia and CO_2 in mobilizing compensatory mechanisms in the body is well recognized (44). Whether distention of the peripheral veins and partial collapse of central veins also initiate reflexes is not known. If the increase in cardiac output during the run (manifested by the increase in pulse pressure and arterio-venous pressure difference) is to be explained on the basis of better heart filling, one must assume either a reflex emptying of blood reservoirs into the circulation, or a constriction of peripheral vessels which displaces blood back to the central veins. The restoration of the respiratory pump will also contribute to circulatory recovery. A typical oscillograph tracing of a run in which pulse pressure and respiration do not return is shown in Figure 9 and one in which both partially recover in Figure 10. The exaggerated pulse pressure following the run may be seen in both cases.

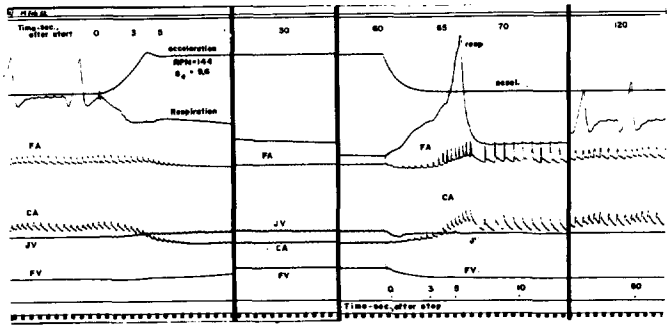

Figure 9 - Recording of physiological parameters in a dog during a tumbling experiment. Center of rotation was through the heart (144 rpm).

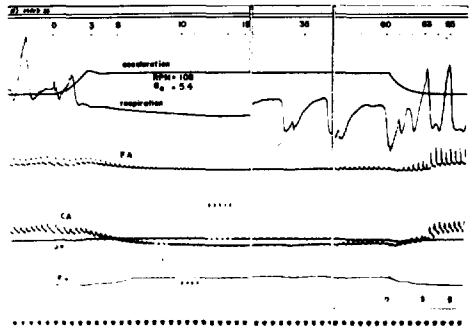

Figure 10 - Recording of physiological parameters in a dog during a tumbling experiment. Center of rotation was through the heart (108 rpm).

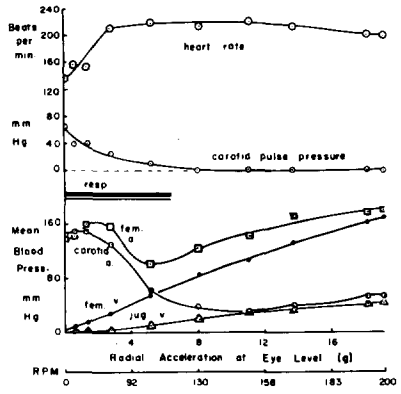

Figure 11 - Effect of spinning a dog about a center of rotation through the heart at various speeds (30 sec.).

Figure 11 shows the various parameters existing at 30 seconds after the start of acceleration plotted as a function of the speed of each run. The values plotted here were obtained from runs on a single animal in order to present a common vascular geometry for the series and are typical of the many other cases. It may be noted that the tachycardia does not increase beyond a table speed of 80 rpm , possibly because the physiological limit of heart rate has been reached. At higher speeds of rotation (i.e., above 170 rpm) the tachycardia is not as high as it is at lower speeds. This may reflect a limitation to heart rate imposed by the decreased coronary supply since, as already noted, (Figures 5 and 6) after 30 seconds of spin at higher speeds the rate has fallen off appreciably. For this animal apnea, loss of pulse, and reduction of the arterio-venous pressure difference to less than 20 mm Hg all occur at about 125 rpm . A compilation of 62 similar runs on 12 dogs is shown in Figure 12, but for the carotid and jugular pressures only. An example of the degree of scatter of the data from which the compilation has been made is presented in Figure 13. From Figure 12 it can be seen that the disappearance of pulse pressure and arterio-venous pressure difference and, by implication, of cardiac output, occurs at speeds between 140 and 150 rpm in the nembutalized dog. Ten seconds of exposure to such conditions are said to result in loss of reflexes (45). The speed of rotation at which the arterio-venous pressure difference falls to 25 mm Hg (approximately 125 rpm) may be an earlier critical endpoint, since Henry et al (46) report that in humans arterio-venous pressure differences below this value result in unconsicousness. As the speed of rotation increases, it is noticed that the arterial pressures which have fallen at lower speeds are now rising. This is of no value to the circulation, however, because the venous pressures are rising simultaneously. Although an increase in arterio-venous pressure difference often occurs at higher speeds, it is probably an artefact, as discussed in Appendix A.

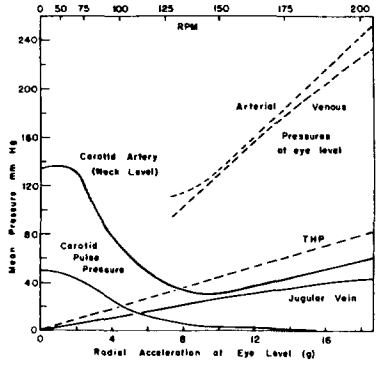

Figure 12 - Relationship of measured and theoretical blood pressures (center of rotation at heart).

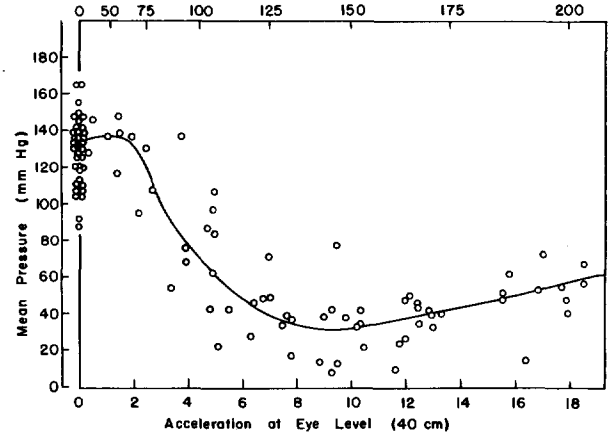

Figure 13 - Carotid pressure during tumbling (center of rotation at heart).

B. Center of Rotation Caudad of the Heart

When the center of rotation is located caudad of the heart, (e.g., 17.5 cm caudad of the ventricle) the effects, as might be expected, resemble those of negative g. Especially noted are the increased hemorrhage and edema in the head region. A group of such spins was run at various speeds and the values of the parameters existing at 30 seconds after the start of the run plotted as a function of the speed (Figure 14). On the same graph are plotted comparable values for runs on the same animal with the center of rotation at the heart (dotted lines). A high speed run (169 rpm) of one minute duration with the center of rotation 13 cm caudad of the heart is shown in Figure 15.

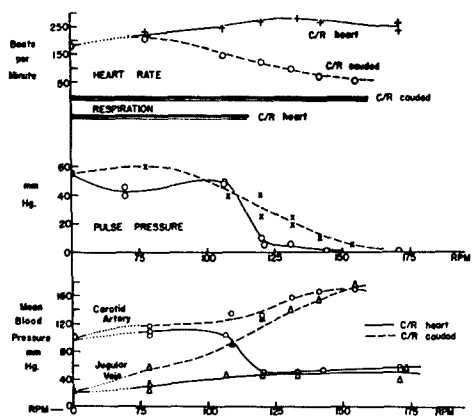

Figure 14 - Effect of change in center of rotation to point (17.5 cm. caudad of heart).

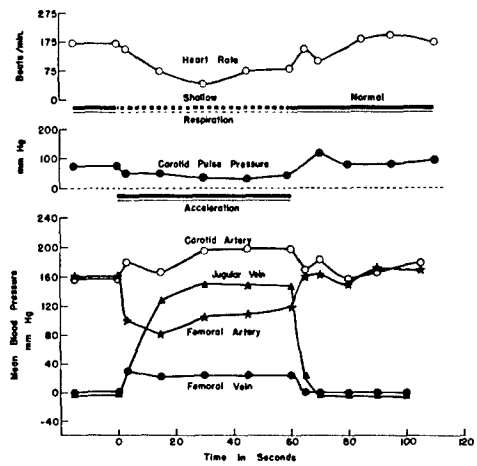

Figure 15 - Effect of spinning a dog about a center of rotation 13 cm. caudad from the heart (169 rpm, 1 min).

Note, in contrast to runs of similar speed with the center of rotation at the heart (Figures 5 and 6) the bradycardia, sustained respiration, the maintained, though reduced, pulse pressure and arterio-venous pressure difference, and greatly elevated jugular pressure. The bradycardia can readily be explained by reference to the carotid pressure. In a spin about a point in the abdomen, the length of the hydrostatic column from the center of rotation to the carotid sinus is much larger than in a spin about the heart. This results in an increase in hydrostatic pressure sufficient to cause a net rise in pressure at the carotid sinus despite reduced cardiac output.

In the case of the caudad center of rotation, respiration, though depressed, persists at higher g-levels probably because the previously mentioned stretch stimulus is not developed. Depression at high speeds may result from pressure of the viscera against the diaphragm.

The carotid-jugular pressure difference decreases mainly as a result of a disproportionate rise in jugular venous pressure. Since the high jugular venous pressure represents hydrostatic pressure which is imposed on the artery to an equal extent, it may be assumed that here too, the reduction in arterio-venous pressure difference reflects a decrease in cardiac output due partly to the bradycardia and partly to venous pooling. It is noteworthy that the arterio-venous difference is higher at a given speed with the center of rotation at the abdomen than at the heart.

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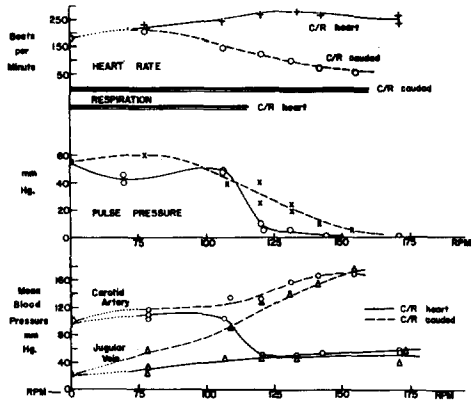
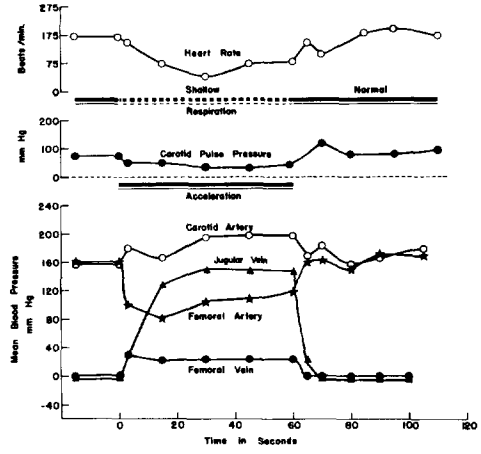


Figure 14 - Effect of change in center of rotation to point (17.5 cm. caudad of heart).



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C. Pressure Relations

The venous pressure at any point during rotations should be roughly equal to the hydrostatic pressure generated by rotation. This pressure may be obtained from the equation derived by Koenen and Ranke (47),

$$P = \frac{\omega^2 r^2}{2}$$

ω = angular velocity

r = distance to the point in question

Since radial acceleration a, at any point $= \omega^2 r$, $P = \frac{a \quad r}{2}$

Thus, if venous pressure at a given radius is plotted as a function of the radial acceleration a straight line should result. Observation of Figure 12 shows this relationship does not strictly hold.

Arterial pressure is the sum of hydrostatic pressure and dynamic pressure resulting from cardiac output. As seen in Figure 12, arterial pressure falls at moderate speeds as a result of the great decrease in cardiac output (and despite the increasing hydrostatic pressure). As the speed increases, the arterial pressure becomes more and more a matter of simple hydrostatic pressure, so that from 150 rpm to 200 rpm it rises almost linearly with radial acceleration. While it would be expected that the arterial and venous pressures would ultimately become purely hydrostatic pressures as the dynamic component contributed by heart action approaches zero, a comparison of the actual carotid and jugular pressures with theoretical hydrostatic pressures (dashed T-H-P line Figure 12) shows a rather surprising result. The measured values are considerably less than predicted. It was first believed that this difference could be the result of a fundamental error in the theoretical equation for estimating expected hydrostatic pressure. Therefore, various lengths of glass tubing open at one end and having a-Statham pressure gage at the other were filled with water and spun at different speeds with the open end located at the center of rotation. Predicted and measured results agreed well within the experimental error (Figure 16) thus indicating it valid to employ the theoretical equation for making such predictions. When the tube was closed at the center of rotation by means of a pressure gage, it was found that a sub-atmospheric pressure developed at the center, and that peripheral pressure could be predicted by adding the value predicted by the equation to this sub-atmospheric pressure at the center (Table I). Furthermore, when tubes closed at both ends and fitted with gages were spun about various points along their length, the same pressure relationship held (Figure 16 and Table I), sub-ambient pressures being developed at the center of rotation. Thus, the equation describes, under any conditions, the pressure difference between any two points in a rotating column of liquid. The discrepancy, then, between measured and predicted blood pressure in the dog experiments may be due to the development of a sub-atmospheric pressure at the center of the column. Alternatively, it may be explained by a partial collapse of the involved vessels such that the length used for making calculations is larger than the length of the filled portion of the column.

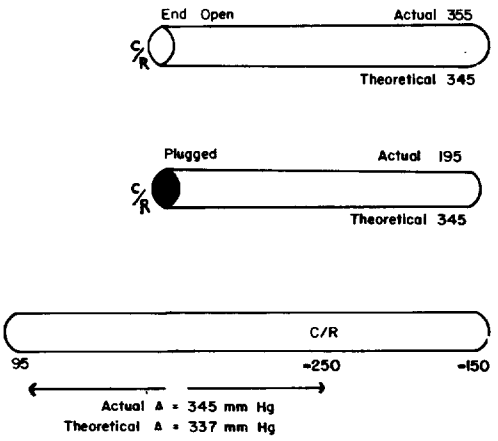


Figure 16 - Behavior of model systems during tumbling. C/R is point at which center of rotation was located in each case.

TABLE I

Comparison of Measured and Calculated Pressures along a Liquid-Filled Glass Tube during Rotation

RPM	Position of C/R	Distance from C/R (cm)	Measured Pressure (mm Hg)	Pressure increment from C/R (mm Hg)	
				Observed	Calculated
162	At end of tube	0	-385	---	---
		24.9	-325	60	66
		37.2	-235	150	149
		55.5	- 55	330	332
		74.8	+210	595	600
163	At center of tube	0	-55	---	---
		12.3	-45	10	16
		37.4	+95	150	153
		18.3	-15	40	36
		37.6	+100	155	154

Evidence to support the view that intravascular pressures considerably below atmospheric are developed at the center of rotation is afforded in two ways; first, by direct measurement, and secondly, by analysis of peripheral pressures. When a catheter is inserted through the carotid artery down to the level of the descending aorta so that its tip is at the center of rotation, it is found that sub-atmospheric pressures develop at speeds beyond 160 rpm. Examples of sub-atmospheric pressures measured in this way in a live animal are offered in Table II. It is true that the polyethylene catheter used was relatively stiff and could be expected not to collapse even under pressure differentials which would surely collapse an artery. However, these measurements agreed well with values calculated simultaneously by an indirect method to be described.

The development of sub-atmospheric pressures at the center of rotation is indicated by a comparison of carotid and femoral arterial pressures when rotation is fast enough to abolish cardiac output. When the theoretical hydrostatic pressure increment from the center of rotation to a point of measurement is subtracted from the pressure measured at that point, the value for the sub-atmospheric pressure, supposedly existing at the center of rotation, can be found. For an unbroken arterial column, the value for central pressure (Pc) should be the same whether estimated from femoral arterial or carotid arterial pressure. In Figure 17 the values of Pc obtained from carotid measurements are plotted as a function of the values of Pc obtained from femoral measurements on the same run. The average length of the carotid column was 22 cm, that of the femoral, 33 cm. If the above approach is valid, the points should fall along a straight line indicating equality of the two estimates, regardless of differences in the lengths of the two columns involved. Although showing considerable scatter, the plot indicates virtual equality of the two values. In addition, statistical treatment of the values of Pc obtained for each run shows there is no significant difference between those obtained from carotid and femoral pressure. For the 22 pairs of values in which the pulse pressure was less than 10 mm Hg (i.e. little or no flow) the "t" value for the differences was 0.05.

TABLE II

Pressures at the Center of Rotation Obtained by an Intra-Aortic Catheter and by Femoral Arterial Measurements

Run #	RPM	Pressure recorded on aortic manometer (mm Hg)	Hydrostatic correction (Distance 23.8 cm)	Calculated pressure in aorta at C/R (Pc)	Pc calculated similarly from simultaneous femoral arterial measurements
4	105	88	-26	+62	+63
3	144	60	-48	+12	+ 9
2	171	50	-68	-18	- 8
1	204	35	-97	-62	-51
5	204	30	-97	-67	-61

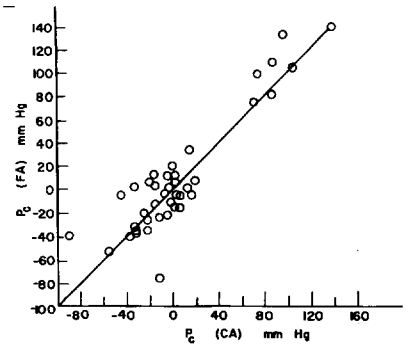


Figure 17 - Comparison of pressure at center of rotation as calculated from femoral and carotid arterial pressures.

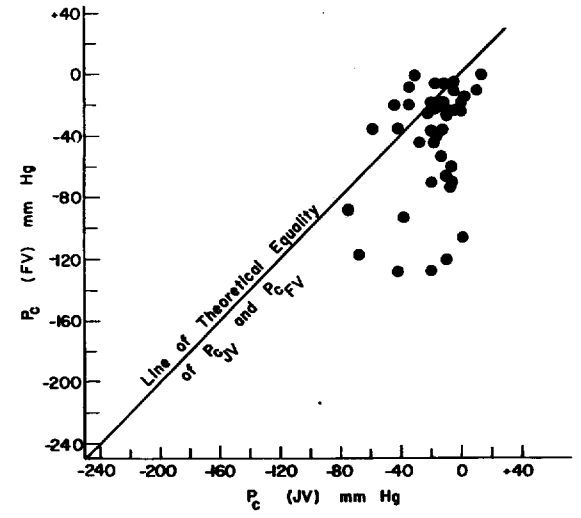


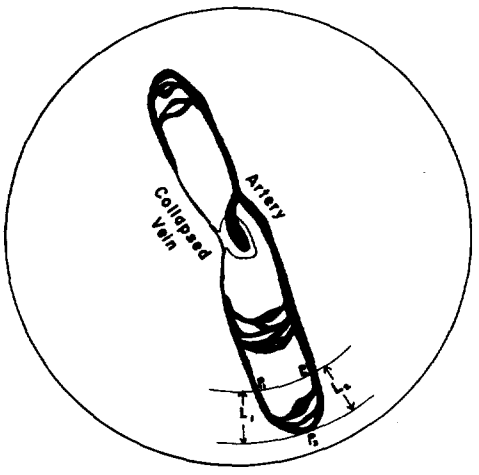
Figure 18 - Comparison of pressure at center of rotation as calculated from femoral and jugular venous pressures.

In the face of a pressure differential of 60 to 70 mm Hg across the walls one might question whether there is sufficient rigidity in the walls of the arteries to remain open at all but it is believed the surrounding tissues behave, in part, as a liquid column and may themselves generate subatmospheric pressures so that the gradient across the wall would be much less than 30 mm Hg. Furthermore, subatmospheric pressures have been previously observed in the head during positive acceleration (46, 48), and have been implied in the work of Clark, Hooker and Weed (49) on venous pressure.

Unlike the situation existing in the arteries, a similar plot of central venous pressures derived from jugular and femoral pressures does not indicate equality of the two values (Figure 18). The points are grouped around the line of equality down to the range at which Pc becomes lower than 20 mm Hg subambient. At this point, the values of Pc calculated from femoral vein measurements fall far too low in comparison to those from jugular vein measurement. If flow has virtually ceased, lack of agreement implies an interrupted venous column and, as might be expected, the longer column to the femoral vein shows a greater collapse.

An important implication of the development of subambient central pressures is that peripheral intravascular pressures produced during rotation will be substantially lower than those predicted by Hill and Shaw (32) and, therefore, tolerances will be higher.

If one considers a vascular system with no flow, it seems that the capillaries should unite the arterial and venous trunks into a U-tube so that at any given distance from the C/R, there would be equal arterial and venous pressures (Figure 19). This would require that values for Pc, even for a partially collapsed venous trunk, should be the same whether calculated from femoral or jugular measurements, provided corresponding arterial extrapolations from each end agree; a situation not found experimentally. The U-tube condition would further require that in a non-flowing system there be no A-V pressure differential, whereas, in fact, the A-V pressure difference persisted and even increased at higher speeds (Figure 12).



$P_1 = P_3 - f(L_1)$ Pressure Effects
$P_2 = P_3 - f(L_2)$ During Tumbling
$L_1 = L_2 \therefore P_1 = P_2$

Figure 19 - Hydrostatics of vascular column during rotation about the heart

These discrepancies may imply either that the arterial and venous trunks behaved as separate systems, or that some flow persisted. In favor of the argument that the U-tube did not exist, is the knowledge that the pressure measurements were made by end catheterization which apparently had the effect of breaking the circulation into separate arterial and venous segments, each with its independent hydrostatic pressure. On the other hand, the fact that an A-V pressure difference persisted throughout the high speed runs, even when pressures were taken in a manner to leave the circulation continuous (Appendix A), certainly suggests that some flow may have continued. This flow may even continue in the presence of a collapsed central venous system (50). The collapse of portions of the venous system would not preclude this flow.

D. Pathology

Hemorrhage may result from over-distension of peripheral vessels as a result of increased hydrostatic pressure. The pathology in the head region resulting from tumbling should be similar to that for negative-g exposures (4, 7, 51, 52), and any pathology caudad of the center of rotation should resemble that of positive g (4, 13). It may be supposed that for a given body region,

the degree of hemorrhage resulting from the tumbling should be of the same order as that resulting from a linear g-exposure which produces the same venous pressure in that area. Post-mortem examinations of dogs were, in most cases, performed immediately after the last of a series of spins. Sacrifice was accomplished by means of an overdose of nembutal and the autopsy performed by a pathologist. In four cases the brain was perfused with one liter of 5% formalin in 0.9% NaCl solution solution prior to opening the cranium. Both gross and histological examinations were made; the results are shown in Table III. The principles referred to were qualitatively demonstrated in the experimental results . Frequent nasal, oral and conjunctival petechiae or frank hemorrhages were observed, occasionally associated with rectal petechiae or hemorrhage. The pressures reported earlier in this paper were measured at the level of the neck or the femoral triangle. Extrapolation of these values to the level of the brain, eye or nasal passages indicates that very high intravenous pressures develop in these areas (Figure 12). This is partially substantiated by the appearance of hemorrhages in the extracranial tissues. For example, subconjunctival hemorrhages were commonly found at a speed of 180 rpm. The venous pressure at eye level associated with this speed was 115 mm Hg.

TABLE III

Summary of Post-Mortem Examination of Eleven Dogs After Tumbling at 195 rpm (1 to 5 runs, 1 minute each)

Organ	Description	Positive Cases	Negative Cases
Brain	Macroscopic Cerebral Hemorrhage	4	7
	Microscopic Cerebral Hemorrhage	5	6
	Hyperemic	6	5
Heart	Subendocardial Hemorrhage	2	9
Lung	Atelectasis	4	7
	Hyperemia or Edema	8	3
	Hemorrhage	2	9
Viscera	Hyperemia	7	4
	Rectal Hemorrhage	1	10

In the case of the brain, the counterpressure exerted by the cerebro-spinal fluid which itself is acted upon by the centrifugal forces, would supposedly prevent rupture (46). The four perfused brains showed no hemorrhage. However, it is difficult to explain the occasional subarachnoid petechiae or the cortical hemorrhages (Figure 20) found as much as one centimeter below the surface upon histological examination. In the first place, these dogs had been exposed to repeated runs in which there was ample opportunity for symptoms of cerebral hypoxia to appear. This condition would lead to weakening of the vessels (7) (53). Secondly, it was observed that systolic pressures as high as 400 mm Hg may be achieved immediately after the run. At this time, contrary to the situation which existed during the run, there is no significant counterpressure being exerted by the cerebro-spinal fluid. The high pressure gradient thus produced across the walls of the small hypoxic vessels may be sufficient to cause rupture. It may be argued that these pressures measured in the carotid artery would be attenuated by the time the

Figure 20 - Hemorrhage in cerebral cortex of dog (ca. 5 mm below surface) after tumbling experiment

wave reached a vessel small enough to rupture. However, as a result of the hypoxic conditions existing during and immediately after the spin, the cerebral arterioles would likely be dilated (54). As a consequence, the attenuation of a pressure wave would be subnormal and sufficient energy might be left to rupture the vessel.

In addition to hemorrhage of varying degrees, there occurred after rotation about the abdomen, a conspicuous degree of engorgement and edema in the head region. The resemblence to negative-g pathology was so marked that no further description will be given here.

The viscera, after rotation at high speed about the heart, showed a general moderate hyperemia. Part of this might be attributed either to the administration of nembutal or to the extended period of inactivation while the animal was lying on one side (often 6 to 7 hours). It is assumed that engorgement of the viscera, due to acceleration would tend to reverse itself before the animal could be sacrificed.

A point of interest was the occasional appearance of subendocardial (or valvular) hemorrhages. These have been known to occur as the result of a forcefully beating empty heart (55). If such a view is valid, these hemorrhages may be offered as additional evidence that cardiac output is drastically reduced during the run.

Following extended runs at speeds above 150 rpm, animals were invariably cyanotic, corroborating the contention that the impaired circulation and respiration was producing widespread hypoxia, if not anoxia. Nevertheless, the dogs generally survived exposures of 2 minutes, even at speeds of 200 rpm. Recovery appeared to be complete within a few days after exposure.

SECTION IV

SUMMARY

Rates of tumbling between 180 and 240 rpm following seat ejection or during long free fall have been reported and may be a source of danger to an escaping crewman. The expected combination of positive and negative-g symptoms may not be analyzed on the basis of current knowledge of linear accelerative effects alone. Consequently, the physiology of tumbling was investigated on dogs as a preliminary to human experiments. Tumbling was simulated on an eight foot (diameter) horizontal turntable capable of rotating up to 200 rpm. The most serious experimental restriction imposed by this apparatus was the absence of the decelerative forces normally expected to be superimposed on the tumbling in an actual ejection.

The centrifugal forces, produced by head over heels tumbling, drive blood toward the extremities resulting in peripheral pooling and decreased heart filling. When anesthetized dogs are spun about the heart at speeds above 140 rpm, cardiac output virtually ceases and acute hypoxia ensues. The A-V pressure difference falls to between 5 and 20 mm Hg, respiration is inhibited, tachycardia develops, and oral, ocular, and rectal hemorrhages may occur. However, animals generally survive 2 to 3 minute exposures to these conditions. The blood pressure pattern is complex in that hydrostatic pressure increases as the square of the distance from the center of rotation while subambient pressures develop at the center of rotation. At 140 rpm, for example,

venous and arterial pressures at eye level are of the order of 120 to 140 mm Hg, but at the carotid sinus level they range between 50 to 70 mm Hg. Thus a tachycardia induced by the carotid sinus reflexes is possible in the presence of oral and ocular petechiae. Upon cessation of spin, the sudden return of cardiac output is associated with systolic pressures as high as 400 mm Hg. Cerebral hemorrhages found in five out of eleven autopsied animals are thought to be related more to these high post-run pressures than to any pressures developed during the tumbling. Moving the center of rotation caudad aids in maintaining circulation during the spin but pressures in the head region are higher because of the longer column of blood being accelerated.

SECTION V

APPENDIX

Comparison of End and Lateral Blood Pressure Measuring Techniques

A. Introduction

The grossly abnormal vascular flow and pressure pattern observed during tumbling raised the question as to whether the end-catheter method of measuring blood pressure was introducing any serious systematic error in the results. In particular, the relative merits of end and lateral pressure which may differ significantly (56 a) were considered. With the conventional end catheters, the kinetic energy associated with friction and flow is converted to pressure whereas for an exact measure of blood pressure, only the potential energy of the blood, i.e., the lateral pressure against the walls of the vessel, is desired (56 b). Although with normal flow the kinetic energy correction is thought to be negligible (56 c), this factor may amount to 5 to 15% of the total energy under some conditions (57 a) (40). Fifteen percent of the peak systolic pressures found following some high speed spins would involve corrections of the order of 60 mm Hg. End systolic pressures as much as 40 mm Hg higher than lateral pressures have been found in the femoral arteries of dogs with aortic regurgitation (58). Differences of this magnitude, especially if exaggerated by accelerative stress, could possibly be significant in the interpretation of the circulatory changes associated with rapid tumbling. Therefore, it was thought desirable to intersperse, wherever possible in the regular series of runs, a number of tests that would help evaluate the differences between end and lateral pressures during all phases of the tumbling experiments. Beckman (5) has made similar end-lateral comparisons in negative g studies in an effort to eliminate "artefactitious" extremes in arterial pressure records.

B. Methods

A common method of measuring lateral pressure in small animals is by means of T-tubes. However, the use of T-tubes involves a disadvantage in that any difference between the internal diameter of the T and the vessel will change linear blood velocity causing the measured lateral pressure to be different from the true lateral pressure. The difficulty in obtaining or constructing T-tubes that accurately matched the blood vessel led to the design and development of a new type of lateral catheter. This device clamps onto the walls of the vessel with, it is believed, a minimum of distortion of flow in the area of insertion.

The lateral catheter is depicted in Figures 21 and 22, and the technique and equipment used in inserting it into the vessel are shown in Figures 22, 23 and 24. A pair of bow dividers is used to puncture the vessel and spread the opening for insertion of the lower, or inner flanged end of the catheter. After insertion, the dividers are removed and the vessel permitted to close over the inner flange and the outer flange, gently but firmly clamped down by means of the lock nut. The scissors clamp, shown in Figure 22, aids considerably in holding the catheter in place while the lock nut is tightened. With a little practice in the technique, firm, leak-proof junctions are quickly produced. The inner tube and flange are coated with silicone before using to inhibit clotting. After the catheter is in place, one or two ligatures to the body wall maintain it at right angles to the vessel.

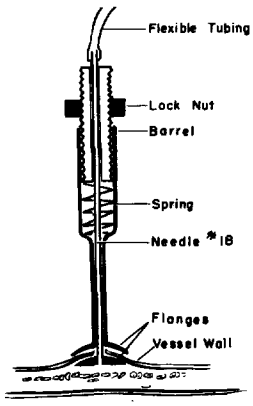


Figure 21 - Diagram of lateral catheter showing principle of fastening to arterial wall.

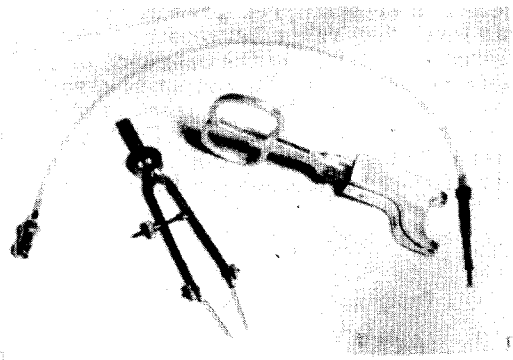


Figure 22 - Bow dividers and scissors clamp used in fastening lateral catheter (also shown) to vessel wall.

Figure 23 - Bow dividers in vessel wall preparatory to spreading opening for insertion of lateral catheter.

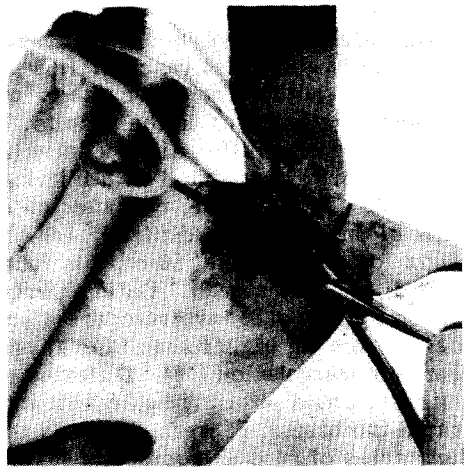


Figure 24 - Lateral catheter in place in arterial wall. The adjacent vein has been catheterized with a polyethylene T-tube.

At the end of the experiment, attempts were made to tie off the insertion opening without blocking the vessel. Tension was applied to the catheter to raise a small nipple on the vessel and a purse string suture tied below the flanges. Unfortunately, in approximately 50% of such ties, the animal hemorrhaged in 24 to 48 hours; apparently this was due to the pulsating pressure working the ligature off the nipple. In later tests, the vessel was ligated on each side of the catheter before its removal.

For purposes of comparison, both lateral catheters and T-tubes were used in some tests. The T-tubes were made of polyethylene tubing by gently warming and pressing together pieces of appropriate diameter and length, a hole having been first bored in the cross piece where the stem was to be attached. Since the use of a lateral catheter was not feasible on thin walled vessels, T-tubes were generally used in the veins if a completely intact circulation was desired. As venous blood velocity is considerably lower than arterial, unmatched diameters in the veins should have far less effect on the kinetic factors. To obtain end pressures with either T-tubes or the lateral catheter, a bulldog clamp was applied to the vessel just distal to the catheter insertion.

As a control check on the functioning of the T-tubes and the lateral catheter in normal animals, a series of static tests were run on a number of dogs before any tumbling occurred. In conducting these tests, the bulldog clamps were simply removed and replaced a number of times on each vessel while continuous blood pressure tracings were being recorded. The information so obtained was compared with data in the literature, and also subjected to mathematical analysis to determine if kinetic energy and frictional loss could reasonably account for the end-lateral difference. In a dynamic series of tests, animals were exposed to duplicate runs with the vessels either clamped or unclamped. All animals were under nembutal anesthesia.

C. Results and Analysis

Tables IV to VII present the changes in blood pressure resulting from obstructing flow in various blood vessels of the normal, anesthetized dog. For the four dogs in which the lateral catheter was used, carotid systolic end pressure averaged 22 mm Hg higher than the lateral pressure and in two dogs in which T-tubes were used in the carotid, systolic end pressures were 39 mm Hg higher than lateral (Table IV). Since the dogs were otherwise treated the same and had comparable end arterial pressures, the higher end-lateral difference obtained with T-tubes may, in part, reflect velocity changes due to unmatched internal diameters between the T-tubes and the artery. A similar situation was observed in the femoral artery (Table V).

TABLE IV

Comparison of End and Lateral Pressure in the Carotid Artery of the Dog (Static Tests). All values in mm Hg.

Lateral Catheter 4 Dogs	End Pressure		Lateral Pressure		End-Lateral Difference ΔP	
	Average	Range	Average	Range	Average	Range
Systolic Pressure	162	140-186	140	125-155	22	15-31
Diastolic Pressure	122	109-135	109	100-117	13	5-22
Pulse Pressure	40	20-51	31	20-42	10	0-24
Mean Pressure	140	130-169	123	115-134	15	6-35
"T" Tubes 2 Dogs						
Systolic Pressure	164	159-168	125	120-130	39	29-48
Diastolic Pressure	109	105-113	97	89-105	12	0-24
Pulse Pressure	54	54-55	28	25-31	26	24-29
Mean Pressure	134	132-135	111	104-118	23	14-31

TABLE V

Comparison of End and Lateral Pressures in the Femoral Artery of the Dog (Static Tests). All values in mm Hg.

Lateral Catheter 4 Dogs[1]	End Pressure		Lateral Pressure		End-Lateral Difference	
	Average	Range	Average	Range	Average[2]	Range
Systolic Pressure	178	158-198	166	145-186	12 (10)	10-13
Diastolic Pressure	108	96-115	104	93-108	4 (5)	2-7
Pulse Pressure	70	49-87	62	43-82	8 (5)	5-10
Mean Pressure	131	117-142	124	111-135	7 (7)	6-8
"T" Tubes 1 Dog						
Systolic Pressure	168		135		33	
Diastolic Pressure	115		108		7	
Pulse Pressure	53		27		26	
Mean Pressure	134		121		13	

1. Includes one animal with side branch to FA catheterized.
2. Difference obtained from the one animal with the catheter in a side branch to the FA.

The end-lateral pressure difference obtained from the static tests, along with the data reported by Bazett and LaPlace (58) are summarized in Table VIII. Pulse pressures are included in the Table because these authors suggest that an almost direct, linear relationship exists between pulse pressure and the end-lateral difference. The end systolic-diastolic values are listed to give some idea of the comparative circulatory condition of the animals. It will be noted there is some discrepancy between the groups of data, especially if the suggested relationship to pulse pressure is taken into consideration. For example, in the femoral artery, Bazett and LaPlace measured an end-lateral difference of 18 for a pulse pressure of 52, whereas in this investigation, a difference of 12 for a pulse pressure of 70 (lateral catheter) or of 33 for a pulse pressure of 53 (T-tube) were found. As the point of pressure measurement is roughly the same in both cases (2-4 cm distal to Pouparts' or the Inguinal ligament), different frictional losses over varying lengths of femoral artery cannot account for the discrepancy. However, since the inside diameter of the T-tube used by Bazett and LaPlace was admittedly smaller than that of the vessel, but larger than that used in our tests, unmatched diameters may again account for part of the difference.

TABLE VI

The Effect on Arterial and Venous Blood Pressure of Obstructing Flow in an Artery (Static Tests). All Pressures in mm Hg

Vessel in which Flow Obstructed → / Vessel in which Pressure Changes Measured ↓	Carotid Artery End-Lateral Difference			Femoral Artery End-Lateral Difference		
	Number of Dogs	Average	Range	Number of Dogs	Average	Range
Carotid Artery						
Systolic				3	4	+1 to +9
Diastolic		(See Table I)			0	-10 to +10
Pulse					4	-? to +1?
Mean					2	-5 to +11
Femoral Artery						
Systolic	3	4	-3 to +8			
Diastolic		2	-3 to +6		(See Table II)	
Pulse		2	0 to +5			
Mean		2	-3 to +7			
Jugular Vein	3	0	0 to 0	2	0	0 to 0
Femoral Vein	3	0	0 to 0	3	-2	-5 to 0

TABLE VII

Effect on Venous and Arterial Pressures of Obstructing Flow in a Vein (Static Tests). All Pressures in mm Hg.

Vessel in which Flow Obstructed → / Vessel in which Pressure Changes Measured ↓	Jugular Vein End-Lateral Difference			Femoral Vein End-Lateral Difference		
	Number of Dogs	Average	Range	Number of Dogs	Average	Range
Carotid Artery						
Systolic	2	0	0 to 0	3	-1	-3 to 0
Diastolic	2	-2	-3 to 0	3	-2	-3 to 0
Pulse	2	2	0 to +3	3	+1	0 to +2
Mean	2	1	0 to +2	3	-1	-3 to 0
Femoral Artery						
Systolic	2	2	0 to +3	3	-1	-2 to 0
Diastolic	2	2	0 to +5	3	0	-2 to +1
Pulse	2	-1	-2 to 0	3	-1	0 to +1
Mean	2	2	0 to +5	3	0	-2 to +1
Jugular Vein	2	8	0 to +15	2	0	0
Femoral Vein	2	0	0	3	2	0 to +7

TABLE VIII

Summary of End-Lateral Pressure Differences and Calculated Blood Velocities at Systole. Static Tests

Observer	Vessel	Type Catheter	State of Animal	Sys./Dias. (End Pressure) (mm Hg)	End Pulse Pressure (mm Hg)	End-Lateral Difference (P) at Systole (mm Hg)	Length of Vessel[3] (cm) L		Radius of Vessel[3] (cm) r		Calculated Blood Velocity (cm/sec) at Systole
							L_1	L_2	r_1	r_2	
Bazett and Laplace (3)	F.A.	T.T.	Normal Anesth.	158/93	52	18	8.5	1.5	0.15	0.125	77
			Aortic; Regurg.	165/56	109	33					114
Melke and Muller (3)	F.A.	T.T.	Normal Anesth. (?)	179/83	96	33			(?)[2]		
Edelberg et. at.	F.A.	L.C.	Normal Anesth.	178/108	70	12	10		0.15		78
		T.T.		168/115	53	33	8.5	1.5	0.15	0.108	87
	C.A.	L.C.	Normal; Anesth.	162/122	40	22	20		0.15		86
		T.T.		164/109	54	39	18.5	1.5	0.15	0.108	84

1. T.T. = "T" tube, L.C. = lateral catheter.
2. Diameter of "T" tube not given.
3. L and r refer to measurements when the lateral catheter was used; L_1 and L_2, r_1 and r_2 to measurements made when "T" tube was used

From Table VI it is seen that clamping either the carotid or femoral artery had approximately the same small effect on pressures in the unmanipulated artery, a 4 mm Hg rise in systolic and a 2 mm Hg rise in mean pressure. The effect on venous pressure was insignificant. The fact that obstructing flow in the carotid had such a small effect on the systemic arterial pressure would suggest that the carotid sinus played a minor role in the development of the observed end-lateral pressure difference. The apparently negligible role of the carotid sinus was further borne out by the abruptness with which carotid arterial pressure rose and fell, and the uniform level maintained thereafter, when the vessel was clamped or unclamped (Figure 25). The similarity of the systemic blood pressure rises produced by clamping either artery suggested that possibly changes in peripheral resistance may account for the increase. In general, the effect on arterial or venous pressures, of clamping a vein appeared to be negligible, (Table VII).

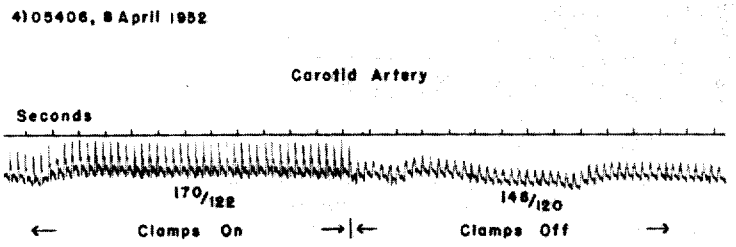


Figure 25 - Changes in pulse form and pressure resulting from conversion of end pressures to lateral pressures by removal of distal clamp

Theoretical treatment of the end-lateral pressure differences, based on the formulae given by Lamport (56b), allowed calculation of flow velocities in the unobstructed vessel. The consistency of these calculated velocities within themselves, regardless of the vessel or type of catheter used, and their agreement with values in the literature can be used to determine if the observed end-lateral differences are valid changes resulting from the conversion of kinetic to potential energy.

The end-lateral pressure difference (ΔP) is the sum of the energy used to overcome friction ($\Delta P'$) and to produce movement ($\Delta P''$), giving

$$\Delta P = \Delta P' + \Delta P'' \qquad 1.$$

$\Delta P'$ can be evaluated by Poiseuille's equation, which appears to apply to whole blood flowing in vessels the size of the major arteries. Thus;

$$\Delta P' = \frac{8 L \lambda v}{r^2} \qquad 2.$$

where; L = length of vessel over which frictional loss occurred (cm)
λ = viscosity of whole blood in poises (g/cm sec)
v = linear velocity of flow (cm/sec) assumed uniform over the length (L)
r = radius of the vessel (cm) assumed uniform over the length (L)

The kinetic energy of motion corrected for laminar flow, is given by:

$$\Delta P'' = \rho v^2 \cong v^2 \qquad 3.$$

where; v = linear velocity at the point of pressure measurement (same as in eq. 2)
ρ = density of blood (g/cm, taken as 1)

Substituting 2 and 3 in 1, expressing all values in cgs units, except that ΔP is in mm Hg, and solving for v by means of the quadratic solution, we get;

$$v = \frac{\frac{-8L\lambda}{r^2} \pm \sqrt{\left(\frac{8L\lambda}{r^2}\right)^2 + 5332\,\Delta P}}{2} \qquad 4.$$

Equation 4 holds only if the radius, and hence the velocity of blood, is the same over the length L, and therefore applies to the lateral catheter data where we have assumed no distortion of the vessel. In the case of the T-tubes with internal diameters not equal to that of the vessel, different velocities will exist in the vessel and the cross pieces of the T. The relationship between these velocities may be expressed by;

$$\frac{V_2}{V_1} = \frac{r_1^2}{r_2^2}, \quad V_2 = \frac{V_1 r_1^2}{r_2^2} \qquad 5.$$

where; v_2 = blood velocity in cross-piece of T-tube (cm/sec)
v_1 = blood velocity in remainder of vessel

r_2 = radius of cross-piece of T-tube (cm)
r_1 = radius of vessel

The kinetic energy terms of equation 1 will now be expressed by:

$$\Delta P' = \frac{8L_1 \lambda v_1}{r_1^2} + \frac{8L_2 \lambda v_2}{r_2^2} \qquad 6.$$

and

$$\Delta P'' = \frac{v_1^2 r_1^4}{r_2^4} \qquad 7.$$

where the subscript 1 refers to that portion of the vessel up to the beginning of the T-tube and the subscript 2 to the cross-piece of the T-tube from the proximal end to the stem (usually one half the length of the cross-piece).

Substitution of 6 and 7 in 1, and solving for v, as before, gives;

$$V_1 = \frac{\frac{-8\lambda\,(L_1 r_2^4 + L_2 r_1^4)}{r_1^2 r_2^4} \pm \sqrt{\left[\frac{8\lambda(L_1 r_2^4 + L_2 r_1^4)}{r_1^2 r_2^4}\right]^2 + 5332\left(\frac{r_1^4}{r_2^4}\right)\Delta P}}{2\left(\frac{r_1^4}{r_2^4}\right)} \qquad 8.$$

Only systolic end-lateral pressure differences have been given mathematical treatment here. One reason for this lay in the desire to compare results with available data in the literature which are given in terms of systolic end-lateral differences. More important, however, is the necessity of choosing a single point in the cardiac cycle for solution of Equation 8. The derivation of this equation shows that the non-linear relationship between v and ΔP makes it meaningless to substitute for ΔP the difference between mean end and mean lateral pressures. Substitution of diastolic values indicate the occurrence of back-flow which would even further invalidate the use of mean values. The data needed for substitution in Equations 4 and 8 and the resulting calculated velocities

are listed in Table VIII. The viscosity of dog blood is given in the literature as 0.036 poises (59). Lengths of the vessels involved were based on measurements made at the time of the experiment. On the basis of similar points of catheter insertion, the vessel length used with the data of Bazett and La Place was taken to be the same as in these tests. The radius of each vessel was estimated at the time of insertion of the catheters, and corroborated by data from the literature (57b).

The range in calculated blood flow velocity at systole for the static series including the data of Bazett and LaPlace, and regardless of the vessel used or the type of cannulation, was 77 to 87 cm/sec (Table VIII) showing fairly good agreement considering ΔP varied between 12 and 39 mm Hg. Linear velocity at systole in the aorta, which can be taken to apply also to the carotid and femoral arteries, is given as ranging between 30 and 83 cm/sec (57c), and may be as high as 100 cm/sec even at low cardiac output (60). Thus, the calculated velocities fall in the upper portion of the expected range. It would appear, from the agreement with the literature and from the uniformity of the calculated velocities, that the observed end-lateral pressure differences are valid changes resulting from the conversion of kinetic to potential energy. Further, it may also be tentatively be assumed that the lateral catheter does not distort flow at the point of measurement, since this was the assumption used in deriving the velocity equation applied to the lateral catheter data.

Data are available on carotid artery end-lateral differences on four dogs in the series in which duplicate tumbling runs were made with arterial clamps on or off (Table IX). Only tests in which pulse pressure dropped to 10 mm Hg or less during the run were used in the comparison, as the most pronounced circulatory changes were associated with such experiments. ΔP was determined at three times during these tests; (1) before the run, (2) during the run, generally 30 sec after the start and (3) at the point of highest systolic pressure, immediately after the run.

Average systolic ΔP for the carotid artery before the run and the velocities calculated therefrom, (Table IX) agree roughly with the static test data of Table VIII. The agreement is especially good in the case of the lateral catheter, but the T-tube results diverged somewhat. As each run subjected the animal to considerable circulatory stress which may have had persistent effects, a close agreement with static, unstressed animal data is perhaps fortuitus.

After the run, carotid artery ΔP's are considerably different than before the run. If the lateral catheter measurements are taken as giving true lateral pressure, then peak systolic end pressure after the run averaged 50 mm Hg too high. Presumably with higher systolic end pressures (measured as high as 400 mm Hg) a proportionally higher ΔP would apply. Pulse pressure is affected approximately the same as is systolic, but diastolic ΔP is reversed, lateral pressure averaging 12 mm Hg higher than end pressure. This diastolic reversal may reflect a strong back flow. Mean pressure shows virtually no change between the end and lateral states. Based on lateral catheter measurements, by calculation (Equation 4) the average velocity at systole after the run is 160 cm/sec.

Only two animals were available for femoral artery end-lateral comparisons, and these failed to support the results obtained from the carotid, either before or after the run (Table X). Both before and after the run the femoral ΔP is considerably lower than expected and, in some cases, reversed in direction. This is thought to be due to the procedure in securing the animal for the tumbling tests. The tight ties required on the hind limbs may have acted as tourniquets so that end pressures were probably recorded even when the arterial clamps were removed.

Comparison of clamped versus unclamped carotid pressures obtained during the run in four animals showed an average ΔP of 9 mm Hg. from which a flow velocity of 35 cm/sec would be deduced. This dilemma, especially in view of the indirect evidence for the cessation of flow (Section III), requires additional data to resolve.

TABLE IX

Comparison of End and Lateral Pressure in the Carotid Artery of the Dog taken from Duplicate Runs before and after Spinning (Dynamic Tests) C/R at Heart. All Pressure Values in mm Hg

Lateral Catheter (2 dogs)	Before Run Average (Range)			At Peak Pressure After Run Average (Range)		
	End	Lateral	Difference (ΔP)	End	Lateral	Difference (ΔP)
Systolic	162 (155-170)	142 (142-142)	20 (12-28) *[80]	298 (265-332)	248 (222-275)	50 (43-57) *[160]
Diastolic	122 (118-125)	107 (102-112)	15 (13-16)	126 (100-152)	138 (138)	-12 (-38-14)
Pulse	41 (30-52)	35 (30-40)	6 (0-12)	172 (165-180)	110 (85-136)	62 (44-80)
Mean	136 (135-138)	126 (121-130)	10 (5-17)	178 (155-202)	178 (170-186	0 (-15-14)
"T" Tube (2 dogs)						
Systolic	178 (175-180)	118 (115-120)	60 (55-65) *[111]	285 (260-310)	128 (125-130)	157 (130-185) * [199]
Diastolic	115 (100-130)	89 (77-100)	26 (23-30)	75 (50-100)	75 (70-80)	0 (-20-20)
Pulse	62 (45-80)	29 (20-38)	33 (25-42)	210 (160-260)	52 (50-55)	158 (110-205)
Mean	140 (132-148)	100 (90-110)	40 (38-42)	143 (93-193)	113 (88-138)	30 (5-54)

* Calculated velocity at systole in cm/sec.

TABLE X

Comparison of End and Lateral Pressure in the Femoral Artery of the Dog taken from Duplicate Runs before, during, and after Spinning. (Dynamic Tests). C/R at Heart. All Values in mm Hg.

2 dogs (1 lateral catheter) (1 "T" tube)	Before Run Average (Range)			At Peak Pressure After Run Average (Range)		
	End	Lateral	Difference (ΔP)	End	Lateral	Difference (ΔP)
Systolic	178 (165-190)	177 (162-192)	1 (-2 to +3)	325 (280-370)	322 (272-327)	3 (-2 to +8)
Diastolic	133 (125-140)	122 (115-128)	11 (10 to 12)	118 (105-130)	120 (95-145)	-2 (-15 to +10)
Pulse	45 (40-50)	57 (48-65)	-12 (-8 to +15)	207 (150-165)	203 (128-278)	4 (-13 to +22)
Mean	147 (137-157)	138 (128-149)	9 (8 to 9)	206 (167-145)	190 (170-210)	16 (-3 to +35)

	During Run (30 Sec. after Start) 168-172 rpm					
	End		Lateral		Difference (ΔP)	
	Average	Range	Average	Range	Average	Range
Mean *	88	70-106	85	72-98	?	-2 to +8

* No measurable pulse at these speeds.

Another discrepency, already referred to (Section III) is the persistence of an A-V pressure difference which increases at high speeds (Figure 12). While in some cases this may conceivably be attributed to the cutting off of a vascular column by end cannulation or clamping, the four cases in which lateral cannulation of the carotid and jugular was employed still showed a persistent, though smaller, A-V pressure difference after the clamps were removed, (8 mm Hg as compared to 15 mm Hg with the clamps on). This makes it likely that at least some flow persisted even at these speeds since the A-V difference of 8 mm Hg in the unoccluded system cannot be attributed to a trapped column.

D. Discussion

Having at least tentatively established that the end-lateral pressure difference, obtained with either T-tubes or the lateral catheter, may be reasonably ascribed to the conversion of potential to kinetic energy and that the lateral catheter appears not to distort flow at the point of pressure measurement, we may now assess the magnitude of the errors likely to be caused by the measuring techniques.

In Table XI the end-lateral pressure differences for static and dynamic tests are listed as percent of end pressure. This determines the correction that should be deducted from end pressure to obtain lateral pressure. Based on lateral catheter data, it would appear that a rather uniform correction of approximately -14% may be applied to the carotid artery systolic end pressure over a considerable range of flow velocities (78 to 160 cm/sec) in order to obtain true lateral pressure. The carotid diastolic and mean pressure corrections are smaller (it may be expected that the greatest loss of potential energy would be associated with that portion of the cardiac ejection cycle involving the fastest flow rate) but less uniform (11% to -10%). The pulse-pressure corrections, apparently involving both systolic and diastolic effects, were the largest encountered (15 to 36%), Table XI.

TABLE XI

Percent Decrease in Pressure when Changing from End to Lateral Measurements

	Carotid Artery						Femoral Artery	
	L.C.*			T.T.**				
	Static	Dynamic		Static	Dynamic		Static	
		Before run	After run		Before run	After run	L.C.	T.T.
Systolic	14	12	17	24	34	55	7	20
Diastolic	11	9	-10	11	23	0	4	6
Pulse	25	15	36	48	53	75	11	49
Mean	11	7	0	17	29	21	5	10

* Lateral Catheter ** T-Tube

Only static data are available for the femoral, but presumably the same situation holds before and after the run as in the carotid. Based on lateral catheter results, the corrections that should be applied to femoral end pressures to obtain true lateral pressures are about half as large as for the carotid. This could be related to the fact that frictional loss occurred over only half as great a length of femoral artery (10 cm) as in the carotid artery (20 cm). Although any of these corrections, femoral or carotid, may be too large to ignore if accurate measurement of the potential energy of the blood is desired, it would seem that except possibly for the pulse pressure measurements, the use of end catheters did not seriously distort the analysis of the circulatory effects associated with tumbling as long as rapid flow is maintained. The distortion (due to a "trapped column") that may be introduced by end catheters during acceleration associated with low cardiac outputs has already been mentioned.

If the corrections that should be applied to end pressure were to be assessed by the T-tube results, a far different interpretation of the distortion induced by end catheterization should have been obtained. As shown in Table XI, the corrections based on T-tube data went as high as 75 %, and were, in most cases, at least twice as high as lateral catheter corrections. The cross piece of the T-tube used in these tests was estimated to be only 0.027 cm smaller in radius than the vessel, and had only 1.5 cm of this radius exposed to frictional loss. Thus it would appear that the use of a T-tube which did not accurately match the vessel may introduce more serious errors in the analysis than the use of simple end pressures.

E. Summary of End-Lateral Pressure Differences

The possible errors which end catheterization may introduce into blood pressure measurements made in connection with tumbling experiments on dogs was investigated by means of T-tubes and a new type of lateral catheter. The design and application of the lateral catheter, which attaches to the wall of the vessel with a minimum of distortion of the vessel diameter, is described. Formulae are derived for calculating the velocity of flow at systole from the difference between end and lateral pressure by equating the difference to kinetic energy factors. The uniformity of the calculated velocities at systole in the unstressed dog (77 to 87 cm/sec) despite a wide range in end-lateral pressure difference (12 to 39 mm Hg), and the reasonable agreement of these velocities with data found in the literature, were taken as indicating that the end-lateral difference was, in fact, a measure of the conversion of potential to kinetic energy. The same argument was used to show that the lateral catheter probably does not distort flow at the point of pressure measurement since the velocity formula used was predicted on this assumption. Lateral catheter values may thus be tentatively accepted as measuring true lateral pressure.

Based on lateral catheter data, a maximum correction of 17% was required to reduce carotid end systolic, diastolic or mean pressure down to lateral pressure, whereas the carotid pulse pressure correction went as high as 36%. Limited data on the femoral indicated that the correction for end pressures in this vessel will be approximately one half that in the carotid. Thus, except for pulse pressure, the use of end catheters is not likely to distort the circulatory effects associated with tumbling providing cardiac output is maintained. When cardiac output is sharply reduced, as during high speed tumbling runs, end arterial pressures may be too high, possibly as a result of a trapped column of blood, resulting in excessively large end-lateral and A-V pressure differences. The use of a T-tube with an inner radius estimated to be smaller than that of the vessel by only 0.037 cm, resulted in corrections which were, in most cases, at least twice as high as those found with the lateral catheter. It would appear that the use of T-tubes which do not accurately match the vessel diameter may introduce more serious errors into the analysis than the use of end pressures.

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